specific reaction with the ordinary laboratory technic, as is illustrated in the following case:

Patient.—Max M., laborer, aged 40, Hungarian, came to this country two years ago. He had had no illness of any kind during the last year.

Present Illness.—The patient had been working daily during that period. On April 10 or 11, 1908, he began to feel moderate general malaise. This malaise, however, did not keep him from going to work. On Thursday, April 16, he failed to report for breakfast. Attempts to arouse him were futile. He was removed from the boarding house to the Emergency Hospital. During the first twelve hours of his stay at the hospital he was semiconscious, and complained of excruciating headache. Delirium followed. The abdominal walls were rigid, especially on the right side. On Friday, April 17, the same symptoms with additional opisthotonus were present, and on Saturday the same symptoms with additional development of internal strabismus. The blood serum was now examined for typhoid agglutinins by Dr. George C. Ruhland, of the Milwaukee Health Department, with positive results, the details of which are described below. On Sunday, April 19, there was profound comatose delirium and development of râles of a moderately moist character on the right side and "moderate" albuminuria. Death ensued Monday morning, April 20. (We are indebted for this report to Dr. Alexander McKinley, of the Emergency Hospital.)

Necropsy.—April 20, 1908. (Irrelevant factors are omitted.) The body was still warm, with no development of rigor mortis. There was marked internal strabismus. The scleræ of both eyes showed a diffuse reddening and were studded with discrete petechiæ.

Cranial Viscera.—When the skull was opened the subdural space was seen to be tensely filled with fluid. Puncture of the taut dura caused a gush of a considerable quanity of slightly turbid, sanguinolent, cerebrospinal fluid. The entire convexity of the brain was covered with a more or less thick, fibrinopurulent material, of a greenish-yellow color. Its consistency was greater in the recesses than on the apices of the gyri. The volume of this exudate increased in direct proportion with the blood supply. It was, therefore, most copious at the base of the brain. There was intense congestion of the pia mater. The brain substance, proper, showed little involvement to the naked eye. The coverings of the spinal cord were in a similar condition, but to a slighter degree. Microscopic examination of the smears showed numerous Gram-negative, intracellular and extracellular diplococci, resembling, to a certain degree, the gonococcus. There were no cells, however, that contained more than two pairs of the micro-organisms, the Diplococcus intracellularis of meningitis. Careful scrutiny of the entrance points of the blood vessels into the cranial cavity did not reveal the avenue of infection.

Abdominal Viscera: The peritoneum was smooth and glistening; the omentum covered all of the small intestines. The peritoneal blood vessels were negative. The situs of the abdominal viscera was negative. The bladder was distended with urine and protruded 8 cm. above the symphysis pubis. The mesenteric lymph nodes were just perceptibly increased, in no way approximating the degree of enlargement found in typhoid or convalescence therefrom. The spleen measured 10 by 6.5 by 3 cm. It was negative. The liver was slightly increased in size. It cut smoothly and was of a brownish-purple color. The hepatic veins were patent and distended; there was venous congestion. Both kidneys showed normal dimensions and were otherwise negative.

Thoracic Viscera: Easily soluble, fibrous adhesions were found in the right pleural cavity, involving the upper and middle lobe of the lung. The lung itself, however, crepitated throughout and floated well above the surface of the water. The middle and lower lobes of the lung contained a smaller amount of air and showed numerous areas of peribronchial reddening and consolidation. The bronchi showed a deeply reddened and swollen mucosa, especially in their narrower ramifications. Similar conditions prevailed in the left lung. Microscopy of the lung tissue revealed a bronchopneumonia, due to aspiration of infected material, very probably during unconsciousness. The bacteriology shows a rather variegated flora of micro-organisms; no pneumococci of Fraenkel and no intracellular diplococci.

Agglutination Test.—In applying the agglutination test, a dilution of the serum of 1 to 100 was used, on a typhoid culture in bouillon not over twenty-four hours old. Positive agglutination was complete within twenty minutes. The culture was a transfer from a regular laboratory stock culture and by no means too attenuated, nor can any fault be ascribed to the physiologic salt solution used in making the dilutions.

We feel convinced of the reliableness of our results from the report made by Symmers and Wilson,* whose findings are identical in the history, postmortem appearances and the agglutination tests, except that in the case reported by them the Widal reaction was obtained in even a dilution of 1 to 200 within fifteen minutes.

We are aware, of course, of the fact that while agglutinins may be nearly, if not quite, specific in their action, a serum which produces agglutination may be far from being so; also, that such agglutinins may be produced by micro-organisms which we do not ordinarily regard as being related, as, for instance, certain varieties of proteus and staphylococcus, which, according to Stern, produce sera of marked agglutinating power for typhoid bacilli also.

While we are almost certain that the specific agglutinins of typhoid and for the *Diplococcus intracellularis* are quite distinct, and that higher dilutions of the serum of this patient would have ultimately agglutinated only with the meningococcus, yet the case appeared to us of sufficient interest for practical purposes to indicate the desirability of its publication here.

It might be added, in support of the diagnosis of the epidemic character of the case, that simultaneously with this one another inhabitant of the same boarding-house was taken with a disease presenting the same symptom-complex and died a day previous at the same hospital. The physician who performed the necropsy did not enter the cranium, and reported the case as ptomain poisoning.

1030 Grand Avenue.

TOXIC EFFECTS FROM BISMUTH SUBNITRATE

WITH REPORTS OF CASES TO DATE

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Toxic effects from the use of bismuth subnitrate in medicine and surgery are so rare that until recently physicians have regarded its administration as perfectly harmless. Schuler[1] and Bardeleben[2] have pronounced its action non-toxic. The latter treated 100 extensive burns by dusting them with bismuth subnitrate and observed no unpleasant symptoms therefrom. Professor Mühlig[3] administered 20 gm. daily for a prolonged period without producing any poisonous effect.

It was, therefore, not surprising that when radiographers began to use bismuth subnitrate for the purpose of obtaining radiographs of the stomach and intestines, they had no hesitancy in administering large doses, and boasted of the safety with which as much as 40 gm. in one dose could be administered.

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* Symmers, W. S., and Wilson, W. J.: Epidemic Cerebrospinal Meningitis with Typhoid Agglutinins in the Blood, Brit. Med. Jour., Sept. 21, 1907.

[1] Ztschr. f. Chir., 1885, cited from Novak.

[2] Deutsch. med. Wchnschr., 1892, No. 23, p. 544.

[3] München. med. Wchnschr., 1901, No. 15, p. 592.

Cases of poisoning resulting from the administration of bismuth subnitrate have been reported as early as 1793, but these were thought to be due to the impurities in the drug, such as arsenic. The first authentic report of bismuth poisoning was made by T. Kocher,[4] in 1882, who observed that the insoluble bismuth preparation, when applied to large wound surfaces, was capable of yielding so much bismuth absorption as to produce poisonous effects, characterized by acute stomatitis, black discoloration of the mucous membrane, usually beginning at the border of the teeth but later spreading over the whole mouth, followed by intestinal catarrh, pain and diarrhea, and in severe cases by desquamative nephritis.

Similar cases were reported by Professor Peterson.[5] Thereafter the literature remained silent on the subject until 1901, when Professor Mühlig reported two cases of severe burns in which pure bismuth subnitrate was applied, and severe symptoms of bismuth poisoning appeared two weeks after application. In both cases symptoms disappeared as soon as the wounds were curetted.

A similar case was reported by Dressman,[6] in which an extensive burn of the third degree was dressed with a bismuth salve and the characteristic symptoms appeared three weeks later. While the symptoms abated as soon as the applications were stopped, the blue marks around the teeth remained for six months.

Wordan, Sailor, Pancoast and Davis[7] reported two cases, in which 60 and 120 gm. respectively were administered each in one dose. Both cases developed the characteristic symptoms of bismuth poisoning and both recovered.

The first fatal case directly traceable to the administration of bismuth subnitrate for diagnostic purposes is reported by Bennecke and Hoffman:[8]

> To a three weeks' old child (suffering from enteritis, weak and emaciated), a mixture of 3 gm. of bismuth subnitrate in 100 c.c. of buttermilk was administered by stomach, in order to diagnose a pyloric stenosis by Roentgenogram. Twelve hours later the child developed cyanosis, collapse, and died in three hours. Postmortem revealed bismuth in the bowel and small quantities in the liver and blood. Methemoglobinemia was present.

From the same clinic a similar case was reported by Boehme:[9]

> *Patient.*—A child, one and a half years old, markedly rachitic, artificially fed and marasmic, received a few grams of bismuth subnitrate by stomach for radiographic purposes. The stomach was washed and no symptoms of poisoning appeared. Two days later a few grams of bismuth subnitrate were injected per rectum for same purpose, and again the bismuth was washed out. Three hours later the child was suddenly seized with pain, cried, became cyanotic, pale, skin cool, pulse small, and died in twenty to thirty minutes.
>
> *Autopsy.*—Distinct methemoglobinemia; all the mucous membranes were brownish discolored; there was marked rachitis, dilatation of stomach, with stenosis of pylorus. The colon contained large quantities of black and white bismuth.

Professor Hefter suggested that death might have been caused by nitrite poisoning. The blood and pericardial fluids were tested, and in both nitrites were found. Bismuth could not be detected in the liver or blood. These chemical findings threw new light on both cases and prompted Dr. Boehme to determine the true cause of bismuth subnitrate poisoning. The results of his experiments were as follows:

Boehme proved by test-tube and animal experiments that the feces of children, when in contact with bismuth subnitrate, would liberate nitrites which would be quickly absorbed from the intestine and found in the urine. While methemoglobinemia was not caused by the absorption, it must be assumed that the absorption of larger quantities would produce methemoglobinemia. Experiments proved this to be a fact.

Collishon[10] reports two cases of accidental nitrite poisoning, in which the sodium nitrite instead of the sodium nitrate was given. The symptoms were cyanosis, extreme weakness, and a grayish-blue discoloration of the mucous membrane and the tongue. The symptoms were so severe as to produce collapse, but cleared up after the drug was discontinued.

Binz[11] injected small doses of sodium nitrite into dogs, causing their death in a few hours, with symptoms of gastroenteritis and methemoglobinemia.

Harnack[12] caused the death of a cat within five minutes after the administration of 5 gm. of sodium nitrite. Methemoglobinemia was present.

Routenberg[13] reported a case in which a methemoglobinemia with the usual symptoms of nitrite poisoning followed the rectal injection of 50 gm. of bismuth subnitrite in 400 c.c. of oil of sesame. While Routenberg ascribes the poisoning to the contamination of the oil, the analogy with other similar cases leads one to suspect that it was a case of nitrite poisoning.

A recent report of a fatal nitrite poisoning due to bismuth subnitrate is published by Novak and Gutig:[14]

> *Patient.*—A man, 44 years of age, in July, 1908, received a rectal injection of four tablespoonfuls of bismuth subnitrate suspended in two liters of water, for the purpose of testing an intestinal obstruction.
>
> *Intoxication.*—Soon after the *x*-ray examination the bowels were washed out, and while he showed no symptoms on retiring, early in the morning the nurse discovered a discoloration of the patient's skin. Gradually the patient became grayish-green, his mucous membranes cyanotic, his temperature 40, his pulse 96, his breathing stertorous. No response could be elicited. Venesection revealed blood to be of chocolate color, due to methemoglobinemia. The patient died eighteen hours after the rectal injection of bismuth subnitrate.
>
> *Spectroscopic Examination of Blood.*—A few hours after death this showed that the blood again became normal, the brown color being changed to red, showing that methemoglobinemia had disappeared after death.

Experiments by Novak and Gutig prove that some animals, as, for instance, cats, are very susceptible to bismuth poisoning, while dogs and rabbits tolerate large doses.

It has been proved that nitrites, when formed, may be neutralized. If iodin nitrate be added to feces containing nitrites the latter will disappear in two or three days. Maasen has proved that certain bacteria in the bowel convert the nitrites into ammonia or into nitrogen. It is likely that the bismuth subnitrate ingested always causes a liberation of small quantities of nitrites, which are either absorbed and owing to the small amount cause no toxic symptoms, or are changed in the intestine into ammonia or nitrogen.

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[4] Samml. klin. Vortr. (Volkmann's), 1882, No. 224, p. 1907.

[5] Deutsch. med. Wchnschr., June 20, 1883.

[6] München. med. Wchnschr., 1901, No. 6, p. 238.

[7] Univ. Penn. Bull., 1906, August, 1906, p. 122.

[8] München. med. Wchnschr., 1906, No. 19.

[9] Arch. f. Exper. Path. and Pharmacol., 1907, p. 441.

[10] Deutsch. med. Wchnschr., 1889, No. 41.

[11] Arch. f. exper. Path. u. Pharmakol., xiii.

[12] Arch. f. exper. Path. u. Pharmakol., October, 1908, p. 246.

[13] Berl. klin. Wchnschr., 1906, No. 43, p. 1307.

[14] Berl. klin. Wchnschr., 1908, No. 39, p. 1764.

When, however, large quantities of bismuth subnitrate are given, and the liberation of nitrites is abundant and can not be neutralized quickly, so that large quantities of nitrites are absorbed, typical symptoms of nitrite poisoning will appear. Alcohols and glycerin accelerate the formation of nitrites in the intestines. This suggests a practical point, namely: When a case of nitrite poisoning is encountered alcoholics and glycerin should be withheld from ingestion and some form of iodin should be administered.

From these experiments and reports of fatal cases it must be concluded that the poisonous effects of bismuth subnitrate were not due to the absorption of the metalic bismuth, but to the absorption of nitrites, which caused the methemoglobin to precipitate in the blood. The methemoglobinemia appears to be the factor producing most of the clinical symptoms, the cyanosis, dyspnea, diarrhea and cramps. These symptoms indicate that the sudden change in the blood impairs the internal or tissue respiration, and the patients succumb with symptoms of suffocation.

It appears that the intestine, and especially the sigmoid and the rectum, are the laboratories for the liberation of nitrites. The bacteria in this part of the intestinal tract evidently are the nitrite-splitting factors whereas those in the stomach and small intestine allow the bismuth subnitrate to pass into the large intestine without liberation of larger quantities of nitrites.

In the fatal cases thus far reported the patients were all suffering from intestinal diseases, especially diarrhea or constipation, which suggests that the intestinal putrefaction accelerated the nitric formation and rapid poisoning.

During the past two years I have employed bismuth subnitrate extensively in the treatment of empyema, sinuses and abscess cavities[15] by injecting into the cavities a mixture containing 33 per cent. bismuth subnitrate incorporated in vaselin. The question arose as to what became of the bismuth paste after injection. A study of the subject revealed the fact that if the paste was not discharged from the sinus soon after injection, but was retained, it became encapsulated and absorbed. In non-resilient cavities, as in bone, the mass was gradually penetrated from all sides by fibroblasts and gradually replaced by connective tissue, while in collapsible cavities, such as the pleura, the expanding lung gradually replaced the slowly absorbed bismuth paste. This could be confirmed by taking radiographs of the regions so injected, at certain intervals.

This, then, proves that the bismuth paste is absorbed, and the question arises: How is the bismuth excreted, and is its absorption harmful? These two problems are now being investigated, but as the investigation is not completed, I shall in this paper bring forth only definite practical facts and defer the publication of the theoretical aspect of the investigation until the data obtained warrant definite conclusions.

From the experiments of Harnack,[16] M. M. Bergerett[17] and E. S. Wood[18] it may be concluded that the bismuth is slowly absorbed and slowly eliminated.

Before attempting to answer the question whether the absorption is harmful, it is necessary to decide whether the harmful effects noted in the cases reported are due to the absorption of the liberated nitrites or of the bismuth itself. After comparing the symptoms of the two cases reported by Collishon, which were caused by sodium nitrite poisoning, with those of other authors, caused by the administration of bismuth subnitrate, one can hardly doubt that the source of poisoning is identical and that the nitrites are the factors which are responsible for the sudden and violent attacks of poisoning. This fact is supported by the experiments of Boehme, and Novak and Gutig.

In my experience of two and one-half years with the injection of bismuth vaselin into sinuses and abscess cavities I have not encountered any case in which the train of symptoms would correspond to that of an acute nitrite poisoning, so graphically described by the various authors who have met with these cases. In many cases I have noticed a slight lividity of the mucous membranes and skin and a bluish border at the margin of the teeth; otherwise conditions were perfectly normal. In only one case did I note a distinct ulceration in the mouth, which was characteristic of the bismuth absorption as described by Kocher. In another case, that of an empyema pleuræ, I injected into the pleural cavity 720 gm. of 33 per cent. bismuth paste, which was retained there for six weeks; then the patient developed a desquamative nephritis, albuminuria, rapid loss of previously gained weight and the blue border around the teeth. As soon as the bismuth paste was withdrawn by means of an injection of olive oil and the application of a specially devised suction pump, all the symptoms disappeared and the patient regained his weight in a few weeks.

I desire to record a case referred to me by Dr. Roberts, in which the bismuth injections had caused severe bismuth intoxication, and after the symptoms abated the patient died:

Patient.—R., a lawyer, aged 57, for many years an invalid, had had tuberculosis of the hip since 1896, and after extensive operations retained several sinuses which discharged large quantities of foul pus.

Bismuth Injections.—In March a 33 per cent. bismuth vaselin paste had been injected into the sinuses by Dr. Roberts twenty times in a period of sixty days, the total amount representing about 400 gm. of pure bismuth subnitrate.

Intoxication.—While the patient's general condition improved at first and the sinuses healed, he gradually developed all the signs of typical bismuth intoxication as described by Kocher. The mucous membrane of the mouth and gums became bluish-black with ulcerations, his teeth became loose and his lips edematous. He experienced great thirst, diarrhea, and had nephritis. The symptoms gradually abated, the patient became well enough to resume his work as an attorney, and for two months was active in his vocation. A radiograph taken of his pelvis demonstrated that there were only small quantities of bismuth within the tissues, but they appeared to be disseminated through the entire pelvis.

Later History.—Aug. 1, 1908, the patient fell and injured his wrist, and a large abscess was formed, which was operated on by Dr. Verity. A large area of necrosis followed. From this time the patient's temperature varied from 99 to 101.5. He lost in strength; his urine was loaded with casts and albumin; his heart became weaker and irregular, and he died Aug. 16, 1908.

Autopsy.—This, made by Dr. Gehrman and myself, revealed all organs practically negative except the kidneys, which showed evidences of incipient contraction, the capsule being irregularly depressed, and the intestines, which were lined with a mucous membrane, resembled huckleberry jam in color.

Histologic Examination.—The microscopic examination of tissues from the liver, spleen, kidney, heart muscle and intestine, and the chemical analysis of tissues were made by Dr. Maximilian Herzog. His report is as follows:

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[15] Illinois Med. Jour., April, 1908, xiii, 402; July, 1908, xiv, 38.

[16] Arzneilehre, 1883, p. 383.

[17] Jour. de l'anat., 1873, p. 242.

[18] Tr. Am. Neurol. Assn., 1883, p. 23.

Liver: A number of parenchyma cells showed bile granules in their paraplasm. Whether the latter also contained here and there bismuth could not be definitely decided, since no microchemical reaction for bismuth is known. It appeared, however, that the granules darker than the bile granules which were found frequently in the liver cells were bismuth granules. The latter could first be seen distinctly and beyond doubt in the intralobular capillaries. The dark granules were seen in the lumen of the small vessels, and crowded in fusiform cells, probably the star cells of Kupfer. In the interlobular veins (the terminal branches of the portal vein) bismuth was present to a large extent; it was found in the vascular endothelium and deposited in the form of fine granules on the free surface of the intima. Bismuth was also occasionally found in the small interlobular bile ducts, but the biliary ducts and capillaries were generally collapsed and empty.

Kidneys: The renal tissue showed chronic interstitial changes with hyaline degeneration of a considerable number of glomeruli. A few of the degenerated hyaline spheres contained lime salts. Some tubules contained hyaline casts; here and there the tubular epithelia showed marked cloudy swelling. There was no bismuth present in the renal sections.

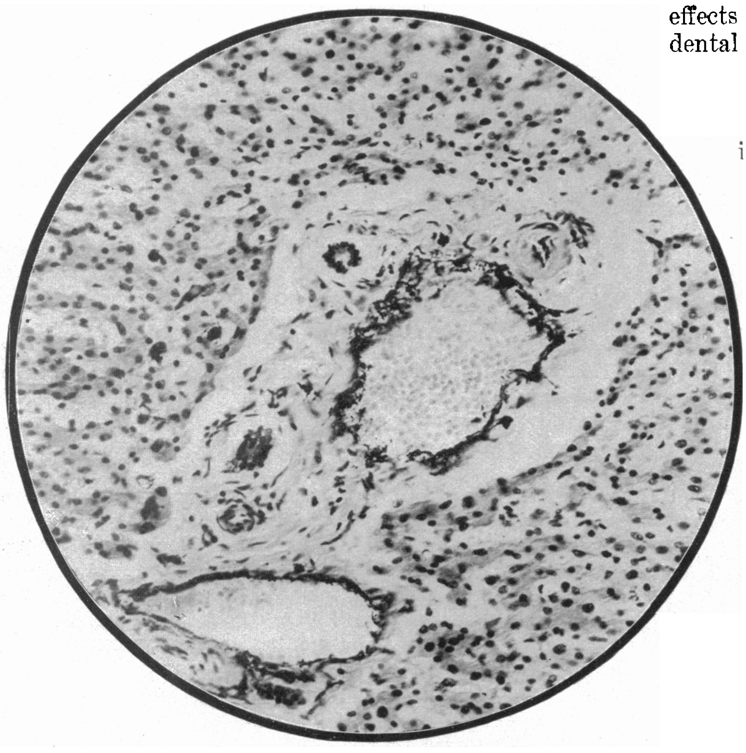

Fig. 1.—Microphotograph of section of liver 4 microns thick. In the center an interlobular vein, to the right and above a sublobular vein. The intima of both lined with bismuth. Magnification, 210 diameters.

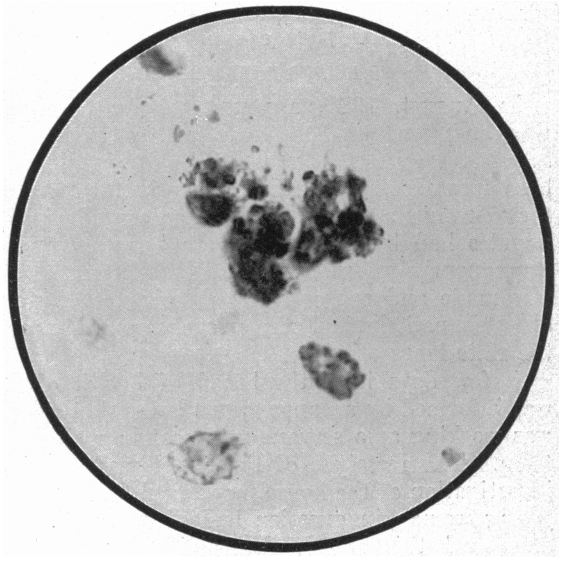

Fig. 2.—Microphotograph of section of liver about 4 microns thick. Some liver cells loaded with bismuth. Magnification, 1,000 diameters.

Spleen: There was some thickening of the capsule and of the trabeculæ noticeable. The pulp spaces were not very distinct and were well crowded with erythrocytes and leucocytes. The Malpighian bodies were not well defined. Some bismuth was present in the shape of amorphous granules and denser masses in the pulp spaces.

Myocardium: Fairly normal.

Intestinal and Lymphoid Tissues: The intestinal tissues showed an extensive infiltration of the mucosa with bismuth. The lymphoid tissue likewise showed bismuth and much of the latter was found in the veins of the submucosa.

Chemical Examination.—The heart muscle contained a faint trace of bismuth. The spleen (very small piece) gave a distinct reaction for bismuth. The liver contained 0.13 per cent. of bismuth oxid. The tissue was pressed fairly dry between filter paper. The total weight of the specimen was 2.292 gm. and the total ash weight 0.03 gm., in which bismuth oxid amounts to 0.003 gm.

The above case does not present the typical symptoms of an acute nitrite poisoning, such as collapse, cyanosis, etc., and while the absorption of the metal bismuth is proved by microscopic examination and chemical analysis, the question still remains whether the absorption and presence of metallic bismuth in the tissues was a cause of death. The pathologic changes in this liver, spleen and heart muscle do not indicate that a severe destructive process which would interfere with function was going on. The interstitial nephritis was probably not due to the bismuth absorption, since the renal tissue was free from deposits of the metal and many pathologic conditions could be expected in an old man who had for years suffered from a chronic suppurative disease. Dr. Verity reports to me that the patient was treated by him ten years ago for chronic nephritis.

The point open to debate is whether this case should go into literature classed as a fatal case of death from bismuth intoxication, or as a case of recovery from the effects of bismuth absorption, death being only accidental and due to other causes.

CONCLUSIONS

1. Bismuth subnitrate administered by stomach in small doses is harmless.
2. In the presence of certain bacteria, or the feces of children, bismuth subnitrate will liberate nitrites, which will be absorbed by the intestines and eliminated by the kidneys; and if the production is faster than the elimination, methemoglobinemia will result.
3. In larger doses per os bismuth subnitrate is liable to produce an acute nitrite poisoning, characterized by cyanosis, collapse, methemoglobinemia, and may terminate fatally.
4. Rectal injection of bismuth subnitrate may cause nitrite poisoning much quicker and more severe than when the drug is administered per os.
5. Children are more susceptible to nitrite poisoning from administration of bismuth subnitrate.
6. Persons suffering with intestinal putrefaction are more susceptible to nitrite poisoning when taking subnitrate of bismuth internally.

7. After the injection of large quantities of bismuth paste into suppurating sinuses, mild symptoms of nitrite intoxication may appear.

8. The bismuth injected into these sinuses and encapsulated will be gradually absorbed and may be found in the liver, spleen, muscles and intestines.

9. Characteristic symptoms of black borders of gums, ulcerations of mucous membranes, diarrhea, desquamative nephritis, may appear several weeks following the injection of the paste.

10. The acute nitrite poisoning is to be regarded as a distinctly separate affection from the more chronic bismuth absorption.

11. Radiographers should employ some other preparation of bismuth instead of the nitrate, and refrain from injections of subnitrate into the bowels, especially if intestinal putrefaction is present.

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A DECEPTIVE CASE OF LEPROSY *

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REPORT BY DR. WHITE

In August, 1907, a young woman from Courland, Russia, was referred to the skin department of the Massachusetts General Hospital for diagnosis. She had been treated one week previously in the "throat room" for "chronic rhinitis" and at her second visit blue-red papules had been discovered on her arm and she was consequently referred to us. The diagnosis of leprosy was made and subsequently corroborated by Dr. Richardson's pathologic findings and the girl was taken to the state leprosorium.

A few days later the board of health of Brookline was informed by an anonymous letter that another leper was working as a servant at a certain residence in that town. The members of the board made an immediate investigation at the house mentioned, but found that the young woman in question had disappeared. Two days afterward this suspected individual presented herself voluntarily at the skin clinic of the Massachusetts General Hospital and was sent to the skin ward for observation and study.

History.—The patient, a young woman, aged 19, was born in a village in the Baltic province of Courland.[1] She stated that her father was dead (cause unknown), but that her mother and brothers were alive and well in their native village. At the age of ten a cutaneous eruption appeared, and on its subsidence scars were found on the right elbow, which still exist. In December, 1906, a sore developed on the genitals and was followed by a rash but no evidence of any labial cicatrix was visible at our first examination. In July, 1907, another outbreak on the skin occurred and was treated by a neighboring physician, but unfortunately his testimony could not be secured. During the last year the girl had been well except for occasional headaches. This was all that could be gleaned from the past history of the case. At entrance on Aug. 24, 1907, the patient stated that the present abnormal conditions began five days previously without any known cause, and she denied absolutely that she had taken any medicines before or since the appearance of her present symptoms.

Examination.—The general condition of the girl was fair; temperature 99.4 F., pulse 90, and respiration 25. The pupils were unequal in diameter and irregular in outline. The conjunctiva of the right eye, especially about the cornea, was considerably injected and pain and photophobia were present. The eyebrows were somewhat scanty, particularly at the distal ends. The tongue protruded straight and was covered with a slight, white coat. Cervical, axillary, epitrochlear and inguinal glands were moderately enlarged and shotty to the touch. The neck was not rigid or tender. Examination of the internal organs revealed nothing abnormal save a soft systolic murmur at the apex of the heart which was transmitted but a short distance toward the axilla. The knee jerks were present and equal. The cutaneous eruption, however, proved more interesting. The face was rather generally suffused and edematous and the nose, in particular, seemed swollen. There were no nodules here, but the lateral diameter was evidently increased and this feature persisted throughout the fourteen weeks of observation. In addition to these general appearances the face, forearms, hands and lower legs showed thickly scattered, round, pea to dime-sized, brick-red macules and maculo-papules with bluish centers. The trunk exhibited a few similar lesions. On the lower legs, especially on the extensor surfaces, more advanced lesions were apparent in the nature of numerous pea to dime-sized exulcerations covered with a sero-purulent, sometimes blackish crust.

Diagnosis.—Despite the anonymous letter and despite the long continued intimacy between this girl and her fellow-townswoman, whom we had so recently adjudged a leper, we were inclined to regard this present eruption as a form of autointoxication resembling erythema multiforme (possibly bullosum).

Treatment.—A soothing carbolic acid and zinc wash, boracic acid ointment, Epsom salts, a diet of milk and soft solids and atropin for the eyes were recommended.

Subsequent History.—On Aug. 27, 1907, three days later, the maculo-papular erythema had perceptibly diminished, the temperature was normal and the crusts had fallen but the ocular symptoms were still present, though to a lesser degree, and on the legs the exulcerations were drying up. Here, however, new features were now exposed consisting of several large pea-sized, round, deep, punched-out ulcers and several still larger pigmented scars. One of these chocolate-colored cicatrices was excised and sent to Dr. Richardson for examination as to the presence or absence of acid-fast bacilli. With these apparently typical gummatous ulcers and the presence of a general adenitis before us, coupled with the history of a previous genital sore, we now made the diagnosis of syphilis and began the tri-weekly injections of a sixth of a grain of bichlorid of mercury which were continued until Sept. 28, 1907.

On Aug. 30, 1907, Dr. Richardson reported the finding of numerous acid-fast bacilli in the sections from the cicatrix of the leg. It must be confessed that this finding was a great surprise, but nevertheless from a clinical standpoint, we were unwilling to believe that the disease was leprosy. Under ether a two-inch incision was made over a subcutaneous mass felt about three inches above the left elbow in the region of the ulnar nerve and two large pea-sized nodules, apparently glands, were dissected out. The nerve itself was found to be perfectly normal. In addition, a small crusted lesion was excised from the right leg, half way between the knee and ankle. An examination of the urine showed it to be slightly cloudy, 1.025 in specific gravity, without albumin or sugar, and containing in its sediment a few round cells, a rare blood globule, but no acid-fast bacilli.

On Aug. 31, 1907, the skin was carefully examined for anesthesia but none was found. The rash had now entirely faded, leaving some pigmentation and a slight, superficial, branny desquamation. The blood showed 100 per cent. of hemoglobin, and a differential count of polymorphonuclears 77

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* Read in the Section on Cutaneous Medicine and Surgery of the American Medical Association, at the Fifty-ninth Annual Session, held in Chicago, June, 1908.

[1] At the Lepra Conference of 1896, it was recorded among other statistics that 134 lepers were known to be living in this province.